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DATE: Wednesday, July 11, 2007

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		DB=PGPB,USPT; PLUR=YES; OP=ADJ	
☐	L8	("20020192163"| "6749834")[URPN]	0
☐	L7	("20020192163"| "6749834")[URPN]	0
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L6	L3 and (angina or infarct or myocardi$ or restenois or heart or cardiac$ or cardio$ or arryth$ or asthma or rectal)	41
☐	L5	L4 and (angina or infarct or myocardi$ or restenois or heart or cardiac$ or cardio$ or arryth$ or asthma or rectal)	70
☐	L4	L1 and (reductant or TCEP or $ethylphosine)	82
☐	L3	L2 and (reductant or TCEP or $ethylphosine)	44
☐	L2	L1 and (thiol or dithiol or dithiothreitol or DTT or "dihydrolipoic acid" or DHLA)	1712
☐	L1	nitroglycerin or GTN or "glyceryl trinitrate"	9550

END OF SEARCH HISTORY

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ACCESSION NUMBER: 1999:524993 BIOSIS
DOCUMENT NUMBER: PREV199900524993
TITLE: Influence of redox compounds on nitrovasodilator-induced relaxations of rat coronary arteries.
AUTHOR(S): Murphy, Michael E. [Reprint author]
CORPORATE SOURCE: Department of Pharmacology and Neuroscience, Albany Medical College, 47 New Scotland Avenue, Albany, NY, 12208-3479, USA
SOURCE: British Journal of Pharmacology, (Sept., 1999) Vol. 128, No. 2, pp. 435-443. print.
CODEN: BJPCBM. ISSN: 0007-1188.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Dec 1999
Last Updated on STN: 5 Jun 2000

AB 1 Various classes of nitrovasodilators release nitric oxide (NO) through distinct reaction pathways, many of which involve endogenous reductants and/or oxidants. This study examined relaxations of isolated rat coronary arteries induced by spermine NONOate (SPNO), 3-morpholinosydnonimine (SIN-1), nitroprusside (NP), S-nitroso-N-acetylpenicillamine (SNAP) and nitroglycerin (NTG) in order to assess whether their potency was influenced by any of six redox compounds: 1 mM ascorbate, 1 mM dehydroascorbate, 0.1 mM dithiothreitol, 10 muM diamide, 0.1 mM ferrocyanide, and 0.1 mM ferricyanide. 2 Only SPNO spontaneously generated NO at measurable levels. These levels were decreased by the presence of ascorbate and dithiothreitol, which likewise decreased the potency of SPNO. 3 The potency of SIN-1 was unaffected by any redox compound except ferricyanide, which increased the potency not only of SIN-1, but also of other nitrovasodilators and NO-independent vasodilators. 4 The potency of NP was decreased by two structurally similar multivalent anions, ferrocyanide and ferricyanide, suggesting that NP metabolism requires ionic binding to tissue. 5 SNAP lost its potency in solutions containing ascorbate or dehydroascorbate. SNAP potency was also decreased by the glutathione oxidant, diamide, and by ferrocyanide and ferricyanide, suggesting that glutathione and ionic binding may be required for NO release. 6 NTG appeared to relax arteries via two pathways. One required only low concentrations of NTG and a labile endogenous factor that was preserved by dithiothreitol and eliminated by ferricyanide. A distinct second pathway required higher concentrations of NTG. 7 These distinct attributes of nitrovasodilator metabolism may underlie differences in regional specificity or tolerance development, and therefore might eventually be exploited in the development and use of nitrovasodilators.

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IT . . .
Circulation); Pharmacology
IT Parts, Structures, & Systems of Organisms

coronary artery: circulatory system

IT Chemicals & Biochemicals
ascorbate; dehydroascorbate; diamide; dithiothreitol; ferricyanide; ferrocyanide; nitric oxide; nitroglycerin: vasodilator-drug; nitroprusside; spermine NONOate: vasodilator-drug; SNAP [S-nitroso-N-acetylpenicillamine]: vasodilator-drug; 3-morpholinosydnonimine: vasodilator-drug

RN 299-36-5 (ascorbate)
10465-78-8 (diamide)
3483-12-3 (dithiothreitol)
13408-62-3 (ferricyanide)
13408-63-4 (ferrocyanide)
10102-43-9 (nitric oxide)
55-63-0 (nitroglycerin)
15078-28-1 (nitroprusside)
136587-13-8 (spermine NONOate)
33876-97-0 (3-morpholinosydnonimine)
79032-48-7 (S-NITROSO-N-ACETYLPENICILLAMINE)

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ACCESSION NUMBER: 1993:323568 BIOSIS
DOCUMENT NUMBER: PREV199396031918
TITLE: Cardioprotective efficiency of dihydrolipoic acid in working rat hearts during hypoxia and reoxygenation: Phosphorus-31 nuclear magnetic resonance investigations.
AUTHOR(S): Assadnazari, H.; Zimmer, G. [Reprint author]; Freisleben, H.-J.; Werk, W.; Leibfritz, D.
CORPORATE SOURCE: Gustav-Embden-Zentrum der Biologischen Chemie, Klinikum der Johann Wolfgang Goethe-Univ., Theodor-Stern-Kai 6, W-6000 Frankfurt/Main 70, Germany
SOURCE: Arzneimittel-Forschung, (1993) Vol. 43, No. 4, pp. 425-432.
CODEN: ARZNAD. ISSN: 0004-4172.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jul 1993
Last Updated on STN: 13 Jul 1993

AB The working rat heart model was used for 31P nuclear magnetic resonance (NMR) studies during normoxia, hypoxia and reoxygenation. Aortic flows of about 35 ml/min could be achieved which equals 65% of the values obtained outside the NMR magnet. Addition of dihydrolipoic acid (DHL) at a concentration of 0.3 mu-mol/l during hypoxia accelerated the recovery of aortic flow and stabilized it during reoxygenation. During hypoxia, inorganic phosphate contents (P-i) were significantly higher in controls. The phosphate shift indicated a pH decrease in control to 6.98, in DHL treated hearts the calculated pH was 7.15. During both hypoxia and reoxygenation, the phosphocreatine (PCr) contents were higher in the DHL treated hearts than in controls. In the controls, saturation transfer measurements revealed a decrease of the flux PCr fwdarw ATP during initial reoxygenation, whereas after addition of 0.3 mu-mol/l of DHL during hypoxia creatine kinase flux remained constant or increased. In isolated rat heart mitochondria, creatine kinase activities were measured under saturating and non-saturating concentrations of PCr. An increase in activity was observed under low PCr (non-saturating) conditions in the presence of 0.7 nmol DHL per mg of protein. At higher concentrations of DHL, creatine kinase activity was increased under all conditions. An increase in ATP synthesis in the working rat heart under influence of DHL is corrobated by NMR spectroscopy.